Formosan Journal of Surgery (2014) 47, 197–200

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CASE REPORT

Complication of an acute subdural hemotoma located between dura and outer membrane space after burr-hole drainage for chronic subdural hemorrhage

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Received 5 December 2013; received in revised form 7 March 2014; accepted 12 May 2014
Available online 1 September 2014

KEYWORDS
burr hole;
postoperative hemorrhage;
rebleeding;
subdural hematoma

Summary Chronic subdural hematoma (SDH) is a common clinical entity in neurosurgery. Craniostomy, including twist-drill holes or burr holes with or without drainage, is the treatment of choice for SDH. Despite convincing results from this surgical procedure, unexpected complications may still develop such as acute epidural hematoma, acute SDH, intracerebral hemorrhage (ICH), tension pneumocephalus, and empyema. We present a rare complication of an acute SDH located between the dura and the outer membrane instead of within the previous hematoma cavity (i.e., between the outer and the inner membrane), which occurred after chronic SDH burr-hole evacuation with closed-system drainage. This complication resulted from misplacement of the drainage catheter and superimposed coagulopathy. In conclusion, precisely inserting the drainage catheter in the chronic SDH space and reversing coagulopathy before and after the surgical procedure are necessary to prevent such complications.

Conflicts of interest: All authors declare no conflicts of interest.

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http://dx.doi.org/10.1016/j.fjs.2014.05.002

1. Introduction

Chronic subdural hemorrhage (SDH) is a common disease in clinical neurosurgery. The incidence is 1—2 cases per 100,000 inhabitants and approximately 13.1 cases per 100,000 inhabitants per year on Awaji Island in Japan.[1]

A review of surgical treatment of chronic SDH suggests that twist-drill craniostomy or burr-hole craniostomy with closed-system drainage is the most effective treatment.[2] Chronic SDH presents favorable results after surgical drainage. Despite this management, recurrent hemorrhage is a major problem. In addition, there have been case reports describing unexpected postoperative complications after chronic SDH drainage such as acute epidural hemorrhage, acute SDH within the previous hematoma cavity, intracranial hematomas, tension pneumocephalus, subdural empyema, and ischemic cerebral infarction.

In this paper, we report a rare case of an acute SDH that developed between the dura and the outer membrane (instead of within the previous hematoma cavity) after burr-hole craniostomy with closed-system drainage. This complication is secondary to the misplacement of the drainage catheter and incomplete reversal of coagulopathy.

2. Case Report

A 55-year-old woman with a history of liver cirrhosis and hepatic encephalopathy (Child classification B) had a motor vehicle accident 3 weeks prior to presentation. Her initial neurological status after the event was clear consciousness with no neurological deficits, except dizziness. However, she complained of having progressive headaches, vomiting, and drowsiness 2 days before she visited our emergency department. Her Glasgow Coma Scale (GCS) score was E2M5V2. Laboratory data revealed a serum ammonia level of 19 mg/dL, platelet count of 85,000/uL, and a mildly prolonged prothrombin time of 13.4/11.9 seconds [international normalized ratio (INR), 1.29]. Head computed tomography (CT) demonstrated bilateral isodense to hyperdense chronic SDH in the frontal, temporal, and parietal regions (Fig. 1).

We reversed the coagulopathy by transfusing 12 units of platelets and four units of fresh frozen plasma (FFP). The platelet count subsequently increased to 119,000 and the INR was 1.1. A bilateral burr-hole craniostomy was performed under general anesthesia with closed-system drainage, and a chronic subdural hematoma, filled with dark red fluid, was evacuated. The procedure was uneventful. She regained consciousness and followed commands immediately after drainage. She was therefore extubated and transferred to the intensive care unit (ICU) for further management. At this point, her GCS was 15 with no focal neurological deficits.

Several hours later, the patient nevertheless suddenly became comatose (her GCS was E1M3V1). The neurological examination revealed a dilated right pupil without pupillary light reflex. The patient was immediately reintubated and reimaged by head CT. The scan image revealed an enclosed residual chronic SDH and additional acute SDH in the right frontotemporal region between the dura and the outer membrane outside the previous chronic SDH (Fig. 2).

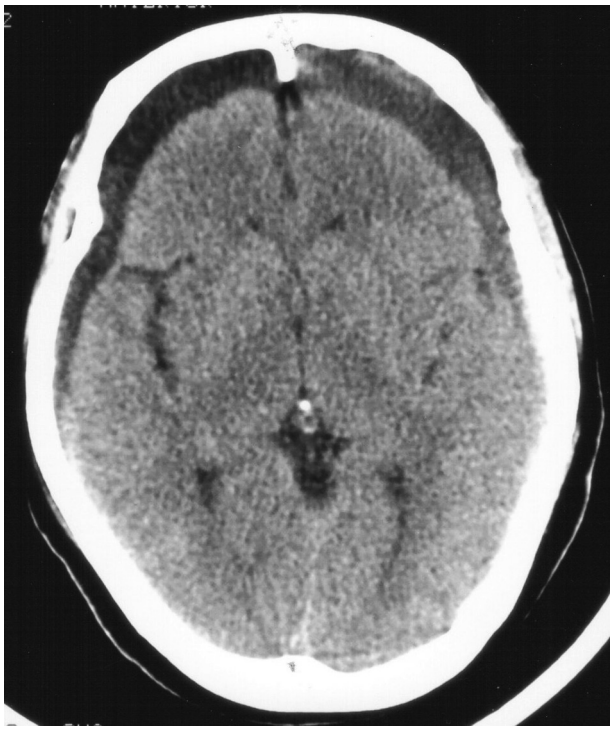

Figure 1 Computed tomography scan of the head shows isodense to hypodense chronic subdural hemorrhage in the bilateral frontal temporal region.

Postoperative coagulopathy was not assessed until her neurological state suddenly altered. These revealed an INR of 1.65 and a platelet count of 78,400. Her coagulopathy immediately reversed with another 12 units of platelets and

Figure 2 Computed tomography scan of the head shows hyperdense hemorrhage at the dura-outer membrane space (thick arrow) in the right frontotemporal region. It is located outside the previous chronic subdural hemorrhage (arrow).

six units of FFP. An emergent right craniotomy was performed. It revealed a fresh clot accumulating between the dura and the outer membrane. This was evacuated with a subsequent outer membranectomy, and the residual chronic SDH was drained. The inner membrane was finally excised as widely as possible to facilitate brain expansion. The procedure was uneventful. Her GCS returned to 15 with full motor power. After the surgical craniotomy, her coagulation data remained within normal limits. Seven days later, she was discharged home with no neurological deficits.

3. Discussion

In 1932, Gardner introduced the osmotic gradient theory as the primary pathophysiology of chronic SDH.[3] This theory suggests that increased protein content in the chronic subdural cavity leads to an influx of fluid as a result of oncotic pressure. The diffusion of fluid across the outer membrane creates a balance between plasma diffusion and/or rebleeding from neomembranes. However, in 1971, Weir[4] disproved this theory on demonstrating that chronic SDH fluid is isosmotic to cerebral spinal fluid (CSF) and blood. Another theory postulates that chronic SDH has a dual origin: one origin is subdural hygroma (SDG) and the other origin is an acute SDH. In an unresolved SDG or acute SDH, the proliferation of dural cells forms an immature neomembrane (i.e., the outer membrane). A repeat microhemorrhage from the fragile new vessels of the neomembrane contributes to the formation of a chronic SDH. When bleeding exceeds absorption beyond the reserve capacity of the cranial cavity, a chronic SDH can enlarge and become symptomatic.

Chronic SDH is a common entity in neurosurgery and has favorable treatment results. However, the definitive treatment strategy remains controversial. Various surgical treatments such as burr-hole drainage, enlarged craniotomy with partial membranectomy and drainage, and extended craniotomy with partial membranectomy with drainage have been retrospectively studied. Simple burr-hole drainage exhibits a lower incidence of rebleeding, compared to the latter two, although all three treatments reveal favorable outcomes. The twist-drill craniostomy or burr-hole craniostomy with or without closed-system drainage is the choice treatment because it is a safe and time-saving procedure and relatively decreases the risk of rebleeding.[5] However, postoperative complications such as acute epidural hemorrhage,[6,7] acute SDH in the previous hematoma cavity,[7] intracranial hematomas,[8] tension pneumocephalus,[9] subdural empyema,[10] and ischemic cerebral infarction remain concerns. The recurrence rate of postoperative chronic SDH is reportedly 2—37%.[11] The most common bleeding site is between the outer and the inner membranes within the original cavity of the chronic SDH.[12]

In this patient, the second head CT image and intraoperative findings confirmed that an acute SDH uniquely developed between the dura and the outer membrane space; this acute SDH was most likely the consequence of a misplaced drainage catheter and underlying coagulopathy.

Misplacing the drainage catheter caused dissection of the potential space between the dura and the outer membrane, and ultimately caused the acute SDH. The small openings from twist-drill holes or burr holes resulted in poor visualization of the subdural space. Every effort nevertheless should be made to ensure the exact position of the drainage catheter into the initial chronic SDH cavity (i.e., between the outer and the inner membranes). A dissection plane between the immature outer membrane and the dura otherwise can result in repeat hemorrhage between the dura and the outer membrane and can result in the rare complication of acute SDH.

Coagulopathy has been identified as a critical contributing factor in the development and recurrence of chronic SDH, acute SDH, and ICH.[8,13] Sixty percent of patients with chronic SDH without a history of trauma were on antiplatelet medication.[14] According to a retrospective study, anticoagulants increase the risk of developing chronic SDH up to 42.5 times, compared to the risk in the general population.[15] When on anticoagulant therapy or in a coagulopathic state, patients with chronic SDH require reversal of the bleeding tendency to prevent the growth of the hematoma and to facilitate the surgical procedure. Patients on dual antiplatelet therapy such as aspirin plus clopidogrel should be paid particular attention.[7] With dual antiplatelet therapy, the platelet count can be normal whereas the prothrombin time (PT) and partial thromboplastin time (PTT) remain unaltered; however, the bleeding time is prolonged with poor hemostasis during the surgical procedure. There is a case report of simultaneous SDH and posterior fossa ICH in a patient using dual antiplatelet medication, which led to mortality following burr-hole drainage.[8] Therefore, the reversal of antiplatelet therapy or coagulopathy prior to surgical drainage is crucial for diminishing the risk of rebleeding.

Our patient's coagulopathy was reversed before surgery, although her coagulation laboratory data were not followed after surgery until she became comatose again. If this were monitored, SDH recurrence presumably could have been avoided in this patient. Therefore, to prevent a high recurrence rate of acute SDH after treatment for chronic SDH in patients with bleeding tendencies, coagulopathy should be monitored and reversed before and after surgery. This is paramount in preventing delayed SDH after the drainage of chronic SDH.

4. Conclusion

Chronic SDH is a common disease in clinical neurosurgery with a favorable prognosis after burr-hole drainage, although complications such as rebleeding remain a problem. Despite the small opening of twist-drill holes or burr holes, every effort should be made to insert the drainage catheter precisely into the original chronic SDH space. Early recognition of patients at risk for bleeding tendencies, effective reversal of coagulopathy, and careful preoperative and postoperative monitoring of coagulation profiles are mandatory to prevent the incidence of SDH.

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